

Contagious Depression: Negative Attachment Cognitions as a Moderator of the Temporal Association Between Parental Depression and Child Depression

John R. Z. Abela, Suzanne Zinck, Shelley Kryger, and Irene Zilber

Department of Psychology, McGill University

Benjamin L. Hankin

Department of Psychology, University of South Carolina

This study examined whether negative attachment cognitions moderate the association between the onset of depressive symptoms in children and their parents using a high-risk sample (parents with a history of major depressive episodes and their children) and a multiwave longitudinal design. During the initial assessment, 140 children (ages 6–14) completed a measure assessing parent-child attachment cognitions. Parents and children also completed measures assessing current level of depressive symptoms. Following the initial assessment, children and parents were contacted every 6 weeks for the next year to complete measures assessing depressive symptoms. The results of hierarchical linear modeling analyses indicated that children who exhibited high levels of negative attachment cognitions reported greater elevations in depressive symptoms following elevations in their parent's level of depressive symptoms than children who exhibited low levels.

There is a wide consensus in the psychological literature that depression runs in families (Goodman & Gotlib, 2002; Hammen, 1991). Previous research has shown that children of parents with a history of major depressive episodes are four to six times more likely than other children to develop major depression (Beardslee, Keller, Lavori, Staley, & Sacks, 1993; Hammen, Burge, Burney, & Adrian, 1990; Weisman, Warner, Wichramaratne, Moreau, & Olfson, 1997). Furthermore, previous research has reported a significant temporal association between mother and child diagnoses (e.g., Hammen, Burge, & Adrian, 1991; Radke-Yarrow, Nottelman, Belmont, & Welsh, 1993). At the same time, relatively

little research has examined potential factors that moderate this association. Clearly not all high-risk children develop depressive symptoms following the onset of parental symptoms. Thus, it is possible that some children possess certain characteristics that make them more vulnerable than other children to the deleterious impact of parental depression. Identifying such vulnerability factors is likely to be beneficial in guiding clinicians and researchers in designing effective treatment and prevention programs for children of depressed parents. Given the high degree of risk for developing depression among such children, particularly following the onset of depressive symptoms in their parents, research in this area is desperately needed.

One framework from which the temporal association between parent and child depression can be examined is from the perspective of Bowlby's (1969, 1980) attachment theory. According to Bowlby, early attachment patterns between children and their caregivers play a vital role in both normal and abnormal development. Attachment patterns are thought to derive primarily from the quality and the quantity of contact that the

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Correspondence should be addressed to John R. Z. Abela, Department of Psychology, McGill University, Stewart Biological Sciences Building, 1205 Dr. Penfield Avenue, Montreal, Quebec, Canada, H3A 1B1. E-mail: john.abela@mcgill.ca

child has with his or her caregivers (Ainsworth, Blehar, Waters, & Wall, 1978). Parents who are sensitive in their caregiving, alert to their infant's needs, and react quickly and appropriately to such needs are likely to have infants who develop a secure attachment (Wenar & Kerig, 2000; West, Spreng, Rose, & Adam, 1999). Attachment theorists hypothesize that the formation of a secure attachment facilitates the subsequent development of trusting and dependable relationships with others and has important consequences for the child's sense of security, adjustment, and emotions.

Not all children, however, develop a secure attachment to their caregivers. When normal developmental processes go awry, a number of types of insecure attachment patterns have been hypothesized to result. Insecure attachment patterns have been posited to serve as vulnerability factors for a diversity of psychological problems including depression (e.g., see Davila, Ramsay, Blum, & Steinberg, 2005). For example, Bowlby (1980) posited that early experiences with interpersonal loss, whether actual or perceived, may increase risk for developing depression—particularly when such loss is perceived as uncontrollable. Bowlby hypothesized that the relationship between such loss and depressive symptoms is mediated by negative internal working models about the self and others. For example, children who fail to form stable and secure attachment relationships, despite persistent attempts to do so, are likely to develop negative self-representations. Similarly, children who receive messages from their parents that they are incompetent or unworthy are likely to develop negative representations of both the self and others (e.g., expecting others to be hostile and rejecting). Such negative internal working models are hypothesized to render individuals vulnerable to interpreting subsequent interpersonal loss as a sign of personal failure. Thus, according to Bowlby, loss of attachment figure(s) exerts its influence on depression, in part, through the development of negative internal working models (Besser & Priel, 2003; Burge et al., 1997; Cole-Detke & Kobak, 1996).

A large body of research has accumulated demonstrating a cross-sectional association between negative attachment cognitions and elevated levels of depressive symptoms in children (Abela, Hankin, et al., 2005; Graham & Easterbrooks, 2000; Muris, Mayer, & Meesters, 2000), adolescents (e.g., Armsden & Greenberg, 1987; Armsden, McCauley, Greenberg, Burke, & Mitchell, 1990; West et al., 1999), and adults (e.g., Carnelley, Pietromonaco, & Jaffe, 1994; Hammen et al., 1995). Far fewer studies, however, have prospectively examined Bowlby's (1980) hypothesis that negative attachment cognitions confer vulnerability to the development of future depressive symptoms. Providing preliminary support for this hypothesis, recent research with university student samples has reported that negative

attachment cognitions predict increases in depressive symptoms over time through the mediating role of dysfunctional attitudes (Hankin, Kassel, & Abela, 2005; Roberts, Gotlib, & Kassel, 1996) and interpersonal stress generation processes (Hankin et al., 2005). In addition, results from a 1-year longitudinal study of female high school seniors have indicated that the relationship between negative attachment cognitions and increases in depressive symptoms over time is moderated by the occurrence of interpersonal stressors (e.g., Hammen et al., 1995). To our knowledge, few studies have prospectively examined the relationship between negative attachment cognitions and depressive symptoms in child and/or early adolescent samples. In addition, no studies have examined whether the relationship between negative attachment cognitions and increases in depressive symptoms over time in youth is moderated by the onset of depressive symptoms in their parents.

For children who exhibit negative attachment cognitions, the onset of parental depression is likely to increase their risk for experiencing depressive symptoms for several reasons (for a more detailed discussion of parental depression as proximal trigger of depressive symptoms in youth, see Hammen, 2002, pp. 176–179). More specifically, parental depression has been found to be associated with higher levels of (1) parental irritability, aggression, dysphoria, and withdrawal (Cohn & Campbell, 1992; Cummings, Zahn-Waxler, & Radke-Yarrow, 1981); (2) dysfunctional parenting practices, such as inconsistent, lax, and ineffective child management (Fendrich, Warner, & Weissman, 1990); and (3) marital conflict (Beach, Smith, & Fincham, 1994). Results from a recent meta-analysis suggest that such interpersonal impairment is more pronounced in families with a currently depressed parent than in families with a parent with a lifetime diagnosis (Lovejoy, Graczyk, O'Hare, & Neuman, 2000) suggesting that such impairment is likely to represent a change from baseline family functioning. Thus, for children who possess negative attachment cognitions, the deleterious impact of parental depression on family functioning is likely to trigger negative representations of the self and others and consequently increase risk for depression.

The goal of our study was to examine whether children who exhibit negative attachment cognitions are more likely than other children to experience increases in depressive symptoms following increases in their parents' levels of depressive symptoms. We utilized a sample of parents with a history of major depressive episodes and their children. Given that a past history of major depressive episodes is one of the best predictors of future depressive episodes (e.g., Belsher & Costello, 1988), the use of such a sample maximized the number of parents who experienced elevations in depressive symptoms during the course of the study. In addition,

given that children of parents with a history of major depressive episodes are four to six times more likely than other children to develop depressive episodes (Beardslee et al., 1993; Hammen et al., 1990; Weisman et al., 1997), the use of such a sample maximized the number of children who experienced elevations in depressive symptoms during the course of the study. Last, many studies have demonstrated that children of depressed parents are more likely to exhibit an insecure attachment than other children. Further, this association generally appears to be stronger when investigated among children whose parents show clinical levels of depression (Lyons-Ruth, Lyubchik, Wolfe, & Bronfman, 2002). Thus, the use of a high-risk sample maximized the number of children who exhibit negative attachment cognitions leading to a more powerful examination of Bowlby's (1980) vulnerability hypothesis.

We also utilized a multiwave longitudinal design in which children and parents' levels of depressive symptoms were assessed at multiple time points over a 1-year follow-up interval. The use of such a design allowed us to take an idiographic, as opposed to a nomothetic, approach toward examining our hypothesis. More specifically, we examined whether the slope of the relationship between parental depressive symptoms and child depressive symptoms *within* children varied *across* children as a function of negative attachment cognitions.

The procedure involved an initial laboratory-based assessment in which children completed measures assessing attachment cognitions. In addition, children and parents completed measures assessing depressive symptoms. The procedure also involved a series of follow-up assessments, every 6 weeks for the next year, in which children and parents completed measures assessing depressive symptoms. We hypothesized that negative attachment related cognitions would be associated with greater elevations in children's level of depressive symptoms following elevations in their parent's level of depressive symptoms.

METHOD

Participants

Participants were recruited through ads placed in local newspapers as well as through posters placed throughout the greater Montreal area (additional details are provided in Abela, Hankin, et al., 2005; Abela, Skitch, Auerbach, & Adams, 2005). Two hundred fifty people responded to these ads. Respondents were invited to participate in a telephone interview during which a diagnostician administered the affective disorders module of the Structured Clinical Interview for the

DSM-IV Axis I (SCID-I; First, Gibbon, Spitzer, & Williams, 2001). One hundred thirty-three parents met criteria for either a current or past major depressive episode and were invited to participate in the study. One hundred two parents decided to participate.

The final sample consisted of 140 children (69 boys and 71 girls) and one of their parents (88 mothers and 14 fathers). Thirty-eight sibling pairs were included in the final sample. Children's ages ranged from 6 to 14 with a median age of 10. Parents' ages ranged from 27 to 53 with a median age of 41. The sample was 84.3% Caucasian. The mother tongue of participants included English (68.7%), French (9.8%), Spanish (2.9%), and other languages (18.6%). At the same time, all of the participants were fluent in English. Of the parents, 14.7% were single, 43.1% were married, 9.8% were separated, and 27.5% were divorced. The median family income ranged from \$30,000 to \$45,000. The highest level of education completed by the parents was an elementary school diploma for 7.8%, a high school diploma for 14.7%, a community college diploma for 39.3%, a bachelor's degree for 22.5%, and a graduate degree for 15.7%.

Procedure

Institutional Review Board approval was obtained prior to beginning the research protocol. Parents completed a consent form and a demographics form. The children were told that their participation was voluntary. All children decided to participate in the study. During the first half of the initial assessment, a research assistant verbally administered the Children's Depression Inventory (CDI; Kovacs, 1981) and the Inventory of Parent and Peer Attachment (Armsden & Greenberg, 1987) to the child. During this time, a diagnostician obtained information regarding the children's current and past depressive symptoms from the parent using the Schedule for Affective Disorders and Schizophrenia for School-Age Children, Present Version (K-SADS; Kaufman, Birmaher, Brent, Rao, & Ryan, 1996). During the second half of the initial assessment, parents completed the Beck Depression Inventory (BDI; Beck, Ward, Mendelson, Mock, & Erbaugh, 1961) while a diagnostician obtained information regarding the child's current and past depressive symptoms from the child using the K-SADS.

The second phase of the study consisted of eight telephone follow-up assessments, occurring every 6 weeks for the subsequent year. At each follow-up, a research assistant verbally administered the CDI to the child and the BDI to the parent. The average number of follow-up assessments completed by participants was 4.79 ($SD = 2.13$). The number of follow-up assessments completed was not significantly associated with the

following Time 1 variables: parental depressive symptoms ($r = .11$, *ns*), children's depressive symptoms ($r = .09$, *ns*), children's age ($r = -.17$, *ns*), children's gender ($r = -.01$, *ns*), parents' gender ($r = .00$), or children's attachment cognitions ($r = .03$, *ns*). The 7 children who did not complete any follow-up assessments did not significantly differ from the 133 children who completed assessments on any Time 1 child or parent variables.

Measures

The SCID-I (First et al., 1995). The SCID-I is a semistructured clinical interview designed to arrive at current and lifetime *DSM-IV* diagnoses. Our study employed the affective disorders module and the psychotic screen. Diagnostic interviewers completed an intensive training program for administering the SCID-I interview and for assigning *DSM-IV* diagnoses. The training program consisted of attending approximately 40 hrs of didactic instruction, listening to audiotaped interviews, conducting practice interviews, and passing regular exams (85% or above). The principal investigator held weekly supervision sessions. The principal investigator also reviewed interviewers' notes and tapes to confirm the presence or absence of a diagnosis. Discrepancies were resolved through consensus meetings and best estimate procedures. The SCID-I yields reliable diagnoses of depressive disorders (Zanarini et al., 2000) and is frequently used in clinical studies of depression in adults. In our study the SCID-I was used only to establish inclusion/exclusion criteria and is not included in any analyses. In all cases, the participating parent met criteria for either a current ($n = 48$) or past ($n = 54$) major depressive episode as assessed using the SCID-I (First, Gibbon, Spitzer, & Williams, 2001).

K-SADS (Kaufman et al., 1996). The K-SADS is a semistructured clinical interview designed to arrive at *DSM-IV* and Research Diagnostic Criteria diagnoses. The K-SADS is administered separately to the child and the parent. A summary diagnosis is based on both sets of information. The K-SADS yields reliable diagnoses of depressive disorders (Chambers et al., 1985) and is used frequently in studies of depression in children. We assessed both current and past history of clinically significant depressive episodes to control for depression history in our prospective analyses. Dichotomous scores, based on *DSM-IV* depression diagnostic criteria, indicate the presence (coded 1) or absence (coded 0) of current or past depressive episodes. Of the children, 34 met criteria for a current (6%; $n = 8$) or past affective disorder (21%; $n = 26$), and 106 had no history of affective disorders.

BDI (Beck et al., 1961). The BDI is a 21-item self-report questionnaire that assesses the severity of depressive symptoms present within the last 2 weeks. Scores on each item range from 0 to 3, with higher scores indicating more severe symptoms. Total scores range from 0 to 63. Comparisons of the BDI with psychiatric rating of depression in clinical populations have shown it to have good concurrent validity (Beck et al., 1961). The BDI has also been found to possess strong internal consistency (Cronbach's $\alpha = .93$; Beck et al., 1961). We obtained alphas ranging from .89 to .93 ($M = .91$) across administrations indicating high internal consistency.

CDI (Kovacs, 1981). The CDI is a 27-item self-report questionnaire that assesses the cognitive, affective, and behavioral symptoms of depression. For each item, children were asked whether it described how they were thinking and feeling in the past week. Total scores on the questionnaire range from 0 to 52. The CDI possesses a high level of internal consistency (Cronbach's $\alpha = .86$; Nelson & Politano, 1990) and distinguishes children with major depressive disorders from nondepressed children (Saylor, Finch, Spirito, & Bennett, 1984). We obtained alphas ranging from .79 to .87 ($M = .83$) across administrations indicating moderate to high internal consistency.

Inventory of parent and peer attachment (IPPA; Armsden & Greenberg, 1987). The IPPA is an 18-item self-report questionnaire that assesses children's perceptions of their relationships with their parents and close friends. Armsden and Greenberg (1987) posited that in children

the "internal working model" of attachment figures may be tapped by assessing the positive affective/cognitive experience of trust in the accessibility and responsiveness of attachment figures and the negative affective/cognitive experiences of anger and/or hopelessness resulting from unresponsive or inconsistently responsive attachment figures. (p. 431)

Consistent with such an argument, the IPPA assesses three broad constructs as they apply to parents and peers: degree of mutual trust (e.g., *My parents trust my judgment*), quality of communication (e.g., *I can count on my parents when I need to get something off my chest*), and degree of anger and alienation (e.g., *My parents don't understand what I'm going through these days*). Children rate items using a 5-point Likert scale. Within each relationship type (parents vs. peers), the three dimensions have been found to be strongly associated with one another (Armsden & Greenberg, 1987) and

therefore are commonly aggregated to form a composite score reflecting security versus insecurity (Crowell, Fraley, & Shaver, 1999). We used the 12 items assessing attachment to parents. Thus, children's total score was equal to the sum of all items with higher scores indicating higher levels of insecure attachment.

In recent years, attachment has been seen as involving two major dimensions: (a) anxiety about abandonment and (b) comfort with closeness (Bartholomew & Shaver, 1998; Brennan, Clark, & Shaver, 1998; Fraley & Shaver, 2000). A factor analysis by Brennan and colleagues (1998) of all nonredundant items from several self-report attachment measures found items to load onto these two dimensions with items from the IPPA subscales of trust and communication loading primarily on the anxiety dimension and items from the IPPA subscale of alienation loading highly on both the anxiety and avoidance dimensions. Thus, IPPA composite scores have been argued to represent individual differences in secure (low anxious, low avoidance) to insecure (high anxious, high avoidance) attachment (see Crowell et al., 1999, for additional discussion of how the IPPA fits into this two-dimension system).

Research utilizing the IPPA with adolescent samples (ages 10–20) shows that the IPPA is internally consistent (Cronbach's alphas for the IPPA range from .86 to .91), has strong test-retest reliability, and exhibits good validity with IPPA scores being associated with numerous theoretically relevant dependent variables (Armsden & Greenberg, 1987; Crowell et al., 1999). For example, adolescents who report higher levels of security in their relation with their parents report lower levels of interparental conflict (Armsden, 1986), higher levels of self-esteem and life satisfaction (Armsden & Greenberg, 1987), greater use of problem solving strategies (Armsden, 1986), more consolidated identity formation (Schultheiss & Blustein, 1994), and lower levels of distress (Kenny & Perez, 1996). Attachment security to parents and peers has been found to predict higher levels of both loneliness and anxious symptoms through the mediating role of social support (Larose & Boivin, 1997).

Although far less research has utilized the IPPA with children (i.e., 6–10), preliminary data support the reliability and validity of IPPA scores in this age group. For example, IPPA scores possess moderate internal consistency (Cronbach's $\alpha = .76$) and test-retest reliability over a 1-year interval ($r = .63$; Abela, Adams, & Hankin, 2005) in younger children. In addition, higher scores on the Parental Attachment subscale have been found to be associated with lower scores on the Warmth, Responsiveness, and Consistency subscales of the Parenting Dimensions Inventory (Abela et al., 2005) in both younger and older children. In our study, coefficient alpha was .78.

RESULTS

Descriptive Data

Means, standard deviations, and correlations between Time 1 measures and children's age and gender, and parents' gender are presented in Table 1. Pearson point biserial correlations between children's/parents' gender and all Time 1 measures are also included in Table 1. Consistent with past research, higher levels of negative attachment cognitions were significantly associated with higher levels of depressive symptoms in both parents and children. In addition, higher levels of depressive symptoms in parents were significantly associated with higher levels of depressive symptoms in their children. Parents' gender was significantly associated with children's levels of depressive symptoms with children with mothers participating in the project reporting higher levels of depressive symptoms than children with fathers participating in the project. At the same time, parents' gender was not significantly associated with either children's attachment cognitions or parents' depressive symptoms. Further, children's age, gender, and depression history were not significantly associated with children's attachment cognitions, children's depressive symptoms, or parental depressive symptoms. Finally, fathers ($M = 18.63$, $SD = 13.96$) and mothers ($M = 15.26$, $SD = 12.18$) did not significantly differ in terms of BDI scores at the time of the initial assessment, $t(135) = 1.02$, *ns*, or at any of the eight follow-up assessments ($p > .21$) They also did not significantly differ in terms of likelihood of exhibiting a current major depressive episode, $\chi^2(1) = 0.21$, *ns*.

As each child completed the CDI at multiple follow-up assessments, each child has his or her own mean level of depressive symptoms (i.e., his or her average CDI score during the follow-up interval) as well as his or

TABLE 1
Means, Standard Deviations, and Pearson Correlations Between Time 1 Measures. Pearson Point Biserial Correlations Between Children's Gender and Time 1 Measures

	1	2	3	4	5	6	7
1. CDI	—						
2. BDI	.26**	—					
3. IPPA	.46***	.24**	—				
4. CHILD AGE	.07	.00	.14	—			
5. CHILD GENDER	-.06	.09	.01	-.12	—		
6. PAST DIAGNOSIS	.06	-.05	.01	.18*	.11	—	
7. PARENT GENDER	.18*	-.02	-.03	.16	.08	.13	—
<i>M</i>	10.08	19.19	27.61	9.82	0.51	0.21	0.87
<i>SD</i>	6.74	12.13	8.08	2.37	0.50	0.41	0.34

Note. CDI = Children's Depression Inventory; BDI = Beck Depression Inventory; IPPA = Inventory for Parent and Peer Attachment, Parental Attachment Cognitions subscale.

* $p < .05$. ** $p < .01$. *** $p < .001$.

her own degree of variation in depressive symptoms during the follow-up interval (i.e., his or her standard deviation on the CDI across administrations). Within-subject means on the CDI ranged from 0.00 to 29.00 (avg. = 7.78; $SD = 5.40$) whereas within-subject standard deviation on the CDI ranged from 0.00 to 12.73 (avg. = 3.05; $SD = 2.13$). With respect to the parents, within-subject means on the BDI ranged from 1.33 to 40.17 (avg. = 16.30; $SD = 8.33$), whereas within-subject standard deviations ranged from 0.00 to 19.40 (avg. 6.47; $SD = 3.75$).

The Vulnerability Hypothesis

To test our hypothesis that children who possess high levels of negative attachment cognitions would report greater elevations in depressive symptoms following increases in parental depression levels than children who have secure attachment, we utilized multilevel modeling. Analyses were carried out using the SAS (version 8.1) MIXED procedure and maximum likelihood estimation. One of the advantages of multilevel modeling is the relaxation of the requirement of balanced “non-missing” data (Kreft & De Leeuw, 1998). Our dependent variable was within-subject fluctuations in CDI scores during the follow-up interval (FU_CDI). As FU_CDI is a within-subject variable, CDI scores were centered at each participant’s mean such that FU_CDI reflects upward or downward fluctuations in a child’s level of depressive symptoms compared to his or her mean level of depressive symptoms. Our primary predictors of FU_CDI were attachment cognitions (IPPA), fluctuation in parental depression symptoms during the follow-up interval (FU_BDI), children’s age (CHILD AGE), and children’s gender (CHILD GENDER). As IPPA and CHILD AGE are between-subject predictors, IPPA scores and CHILD AGE were standardized prior to analyses. As FU_BDI is a within-subject predictor, BDI scores were centered at each participant’s mean prior to analyses such that FU_BDI reflects upward or downward fluctuations in a parent’s depressive symptoms compared to his or her mean depressive symptoms.

When fitting hierarchical linear models, one must specify appropriate mean and covariance structures. It is important to note that mean and covariance structures are not independent of one another. Rather, an appropriate covariance structure is essential to obtain valid inferences for the parameters in the mean structure. Overparametrization of the covariance structure can lead to inefficient estimation and poor assessment of standard errors (Altham, 1984). On the other hand, too much restriction of the covariance structure can lead to invalid inferences when the assumed structure does not hold (Altham, 1984).

In our first set of analyses, we were interested in examining the effects of IPPA, CHILD AGE, CHILD GENDER, and FU_BDI on children’s CDI scores during the follow-up interval. Consequently, in line with Diggle, Liang, and Zeger’s recommendation that one use a “saturated” model for the mean structure while searching for an appropriate covariance structure, we chose a mean structure that included CHILD AGE, CHILD GENDER, IPPA, FU_BDI, and all two-, three- and four-way interactions. Five additional effects were also included in this initial mean structure. First, to control for individual differences in baseline levels of depressive symptoms, both children’s Time 1 CDI scores (T1_CDI) and past history of clinically significant depressive episodes (K-SADS P-DIAG) were included in the model. Second, to control for the potential differential impact of maternal and paternal depression on children’s symptoms, parents’ gender was included in the model. Third, to account for the possible correlation in response variables between siblings from the same family, random effects for children (RE_CHILD; random intercept) nested within families (RE_FAMILY; random intercept) were included in the model. Last, given that FU_BDI is a within-subject predictor whose effect is expected to vary from participant to participant, a random effect for slope (RE_SLOPE) was included in the model.

Commonly used covariance structures in studies in which multiple responses are obtained from the same individual over time (and consequently within-subject residuals over time are likely to be correlated) include compound symmetry, first-order autoregressive, heterogeneous autoregressive, and banded Toeplitz. To select one of these covariance structures for our analyses, we fitted models utilizing each structure and chose the “best” fit based on Akaike information criterion (AIC and AICC) and Schwarz Bayesian criterion (BIC). In all cases, the best fit was a heterogeneous autoregressive structure (ARH[1]). Such a covariance structure indicates two general patterns in CDI scores during the follow-up interval (for further explanation, see Littell, Pendergast, & Natarajan, 2000). First, as the interval between any two follow-up assessments increases, the degree of intercorrelation between children’s CDI scores at these two time points decreases (i.e., CDI scores obtained at follow-up 4 are more strongly associated with CDI scores obtained at follow-ups number 3 and 5 than with CDI scores obtained at follow-ups 2 and 6). Second, the variance in CDI scores across administrations is not constant (i.e., variance in CDI scores is greater at some follow-up assessments than at others).

After choosing the appropriate covariance structure, we next examined the random-effects component of our model. Nonsignificant random-effect parameters were deleted from the model prior to examining the

fixed-effects component. With respect to random effects, RE_CHILD ($p < .0001$) was significant and thus was retained in the model. Although RE_FAMILY was not significant, it was retained in the model to account for any nonindependence of data resulting from the inclusion of siblings in the study. RE_SLOPE, however, was not significant and consequently was deleted from the model prior to examining the fixed effects. With respect to the covariance structure, the ARH[1] parameter ($r = .23$, $p < .05$) was significant and thus was retained in the model.

When examining the fixed effects component of the model, we used a process of backward deletion. More specifically, we first examined the GENDER \times AGE \times IPPA \times FU_BDI interaction. Given that this four-way interaction was not significant it was deleted and the model was re-estimated. We next examined the three-way interactions. None of the three-way interactions were significant. Consequently, they were deleted and the model was reestimated. We next examined the two-way interactions. Nonsignificant two-way interactions were deleted and the model was reestimated prior to reporting the final estimate for our model.

Results with respect to the fixed-effects component of the model are presented in Table 2.¹ Of primary importance, a significant two-way, cross-level interaction emerged between IPPA and FU_BDI. To examine the form of this interaction, the model summarized in Table 2 was used to calculate predicted CDI scores for children possessing either high or low levels of negative attachment cognitions (plus or minus 1.5 *SD*) whose parents were experiencing either low or high levels of depressive symptoms in comparison to their own average level of depressive symptoms (plus or minus 1.5 \times mean within-subject standard deviation). The results are presented in Figure 1. As both FU_CDI and FU_BDI are within-subject variables centered at each participant's mean, slopes are interpreted as the increase in a child's CDI score that would be expected given that his or her parent scored 1 point higher on the BDI.

Analyses were conducted for each IPPA and BDI condition examining whether the slope of the

¹Because of the small number of fathers who participated in our study, we were unable to examine whether PARENT GENDER moderated any of the associations reported in Table 2. At the same time, to ensure that all results reported in Table 2 extend to children of depressed mothers, analyses were rerun including only children with a depressed mother participating in the study ($n = 125$). The direction, magnitude, and significance level of all effects were similar to those reported in Table 2, suggesting that the results can be generalized to children of depressed mothers. In addition, when only children of depressed mothers were included in analyses, CHILD GENDER was not a significant moderator of any of any association reported in Table 2 suggesting that our results extend to both daughters and sons of depressed mothers.

TABLE 2
Attachment Style to Parents Predicting Depressive Symptoms

Predictor	<i>b</i>	<i>SE</i>	<i>F</i>	<i>df</i>
Time 1 CDI	3.62	0.41	78.43***	116
PAST DIAGNOSIS	-0.29	0.84	0.12	116
CHILD AGE	0.59	0.38	2.37	116
CHILD GENDER	-1.04	0.71	2.18	116
PARENT GENDER	-0.91	1.22	0.46	116
IPPA	-0.34	0.41	0.66	116
FU_BDI	0.08	0.02	13.96***	470
IPPA \times BDI	0.06	0.02	8.58**	470

Note. Time 1 CDI = Time 1 Children's Depression Inventory; IPPA = Inventory of Peer and Parental Attachment, Parent Attachment Cognitions subscale; FU_BDI = Parent Beck Depression Inventory Score.

** $p < .01$. *** $p < .001$.

relationship between parental depressive symptoms and child depressive symptoms significantly differed from 0. Analyses indicated that children exhibiting high levels of negative attachment cognitions reported higher levels of depressive symptoms when their parents were experiencing high levels of depressive symptoms than when their parents were experiencing low levels of depressive symptoms, $t(470) = 4.62$, $p < .001$. At the same time, level of depressive symptoms did not vary as a function of level of parental depression for children exhibiting low levels of negative attachment cognitions, $t(470) = -0.35$, *ns*. Planned comparisons of the slopes

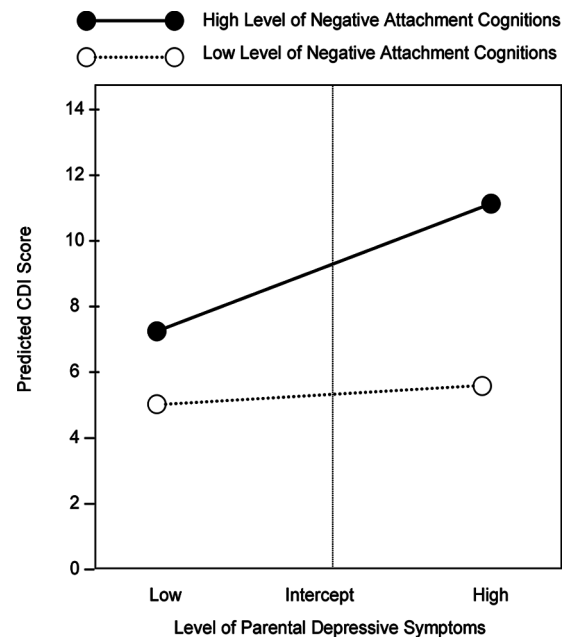


FIGURE 1 Predicted slope of the relationship between parental depressive symptoms and child depressive symptoms for children exhibiting high and low levels of negative attachment cognitions. Note: CDI = Children's Depression Inventory.

of the relationship between parental depression and child depressive symptoms revealed that the slope was significantly greater in children exhibiting high levels of negative attachment cognitions (slope = 0.17) than in children exhibiting low levels of negative attachment cognitions (slope = -0.01), $t(470) = 2.93$, $p < .01$.

DISCUSSION

In line with past research examining the temporal association between parent and child depressive symptoms (e.g., Hammen et al., 1991; Radke-Yarrow et al., 1993), elevations in depressive symptoms in parents during the course of the 1-year follow-up interval were significantly associated with elevations in depressive symptoms in their children. At the same time, expanding on past research, our results suggest that some children may be more vulnerable than other children to the deleterious impact of parental depressive symptoms on children's psychosocial functioning. More specifically, our results suggest that children who possess high levels of negative attachment cognitions are more likely than children who possess low levels of such cognitions to develop depressive symptoms following the onset of depressive symptoms in their parents.

Our results provide support for Bowlby's (1980) vulnerability hypothesis. In line with past research using child, adolescent, and adult samples (e.g., for reviews see Davila, Ramsay, Stroud, & Steinberg, 2005; Hankin & Abela, 2005), children who exhibited high levels of negative attachment cognitions reported higher levels of depressive symptoms than children who reported low levels of such cognitions. In addition, in line with past research using adult samples (e.g., Hankin et al., 2005; Roberts et al., 1996), children's negative attachment cognitions prospectively predicted increases in their levels of depressive symptoms. More specifically, negative attachment cognitions, as assessed at Time 1, interacted with within-subject fluctuations in parental depressive symptoms occurring during the follow-up interval to predict within-subject fluctuations in children's depressive symptoms. As the strength of this association was not moderated by age, our results suggest that negative attachment cognitions confer vulnerability to developing depressive symptoms in both children and early adolescents. This study, to our knowledge, is the first to prospectively examine the relationship between negative attachment cognitions and depressive symptoms in a sample of children and early adolescents. Consequently, our results provide the most powerful support to date for Bowlby's vulnerability hypothesis in a sample of youth.

The findings in our study are consistent with Hammen et al.'s (1995) findings that negative attachment

cognitions confer vulnerability to depressive symptoms following the occurrence of interpersonal stressors (for a more detailed discussion of parental depression as an interpersonal stressor, see Hammen, 2002, pp. 176–179). At the same time, it is important to note that although the onset of parental depression has been conceptualized by theorists and researchers as an interpersonal stressor for children, relatively little is known about children's *actual* perceptions of their parents' symptoms.

Implications for Research, Policy, and Practice

Future research is needed disentangling the ways in which parental depressive symptoms both directly (e.g., making faulty self-attributions for marked changes in parental behavior, assuming greater caretaking responsibilities than are developmentally appropriate, experiencing higher levels of parent-child conflict due to increased parental irritability) and indirectly (e.g., through the role of chronic and/or episodic stressors associated with parental depressive symptoms) impact the stress context of children's lives (Hammen, 2002). In addition, as our study did not examine noninterpersonal stressors, we cannot conclude that negative attachment cognitions confer vulnerability to the development of depressive symptoms *specifically* following the occurrence of negative interpersonal stressors. Future research is needed examining this issue. Finally, future research should also examine additional mechanisms that may account for the temporal association between parent and child depressive symptoms among youth reporting high levels of negative attachment cognitions. For example, increases in parental depressive symptoms may lead to decreases in certain protective factors, such as parental support or supervision, which typically buffer vulnerable youth from experiencing depressive symptoms.

The results of our study have important implications for the treatment and prevention of depression in children of depressed parents. Of interest, despite the fact that a vast body of research has accumulated indicating that children of depressed parents are more likely than other children to develop depression (Beardslee et al., 1993; Hammen et al., 1990; Weisman et al., 1997), relatively little research has focused on developing depression treatment and prevention programs specifically for use with such youth (Gladstone & Beardslee, 2002). Our findings suggest that interventions aimed at building secure parent-child attachment relationships may prove to be effective. Preliminary findings suggest that such interventions can be successfully employed in modifying attachment relationships. More specifically, toddler-parent psychotherapy (TPP; Lieberman, 1992) is a short-term therapy in which the therapist works jointly with mothers and their toddlers in an effort to

build a secure mother–child attachment relationship. Therapy focuses on helping the mother to understand her own attachment history as well as how her history impacts her current relational patterns with her toddler. Emphasis is placed on the therapist building a positive and stable relationship with the mother so that the therapeutic relationship can serve as a working model of a secure attachment relationship (which the mother may have lacked during her own childhood). The assumption is that the mother will utilize the therapeutic relationship as a guide to build a healthy attachment relationship with her toddler. In a preliminary study examining the efficacy of the TPP, Lieberman reported that although prior to intervention, toddlers of depressed mothers were more likely than toddlers of nondepressed mothers to be classified as insecurely attached, postintervention, toddlers of depressed mothers and nondepressed mothers no longer differed in their likelihood of being classified as insecurely attached. Further, postintervention, toddlers of depressed mothers who received the intervention were less likely than toddlers of depressed mothers in the no-treatment control group to be classified as insecurely attached. Although such findings demonstrate that therapeutic interventions may be effective in modifying parent–child attachment relationships, they do not prove that TPP actually reduces risk for future depression in youth. Thus, research is still needed examining the long-term efficacy of TPP in treating and/or preventing depression. At the same time, given that the results of our study suggest that negative attachment cognitions serve as a vulnerability factor to depression in high-risk youth, modifications of parent–child attachment relationships (and consequently modifications of internal working models of the self and the caregiver) may prove to be a promising approach to prevention and treatment in such youth.

Limitations

Several limitations of our study should be noted. First, self-report measures were used to assess depressive symptoms in both the children and their parents. Although the CDI and the BDI both possess high degrees of reliability and validity, it is difficult to make conclusions about clinically significant depression based on self-report questionnaires. Future studies should utilize semistructured diagnostic interviews to see if our findings extend to the onset of clinically significant depressive episodes. Future research should also utilize parent reports of children’s depressive symptoms to examine whether our findings are robust across methodologies (i.e., multi-informant).

Second, our study utilized a sample of high-risk parents (predominantly mothers) and their children.

Although such a design leads to a powerful test of the contagion hypothesis, results can not be generalized to other populations (e.g., samples not recruited specifically on the basis of parental depression). Future research is needed examining whether similar results are obtained using a community sample of children and their parents. In addition, future research should include a greater proportion of fathers to rule out the possibility that the contagion effect observed in our study is limited to mothers and their children.

Third, although our study examined whether negative attachment cognitions moderate the relation between elevations in children’s depressive symptoms following elevations in parental symptoms, future research is needed examining the mechanisms and processes underlying this relationship. Potential mechanisms underlying the deleterious impact of parental depressive symptoms on children’s well-being may include parental withdrawal and irritability (Cohn & Campbell, 1992; Cummings et al., 1981), dysfunctional parenting practices (Fendrich et al., 1990), and marital conflict (Beach et al., 1994). In addition, future research is needed examining the factors that mediate this relationship. Research with adult samples provides support for negative cognitions (Hankin et al., 2005; Roberts et al., 1996) and stress generation processes (Hankin et al., 2005) as possible mediators.

Last, our study examined only the relationship between negative attachment cognitions, parental depressive symptoms, and child depressive symptoms. Thus, we were unable to identify whether the interaction between negative attachment cognitions and parental depressive symptoms specifically predicts the onset of child depressive symptoms or whether it also predicts the onset of other types of symptomatology (e.g., anxious symptoms, externalizing disorders, etc.). Similarly, we were unable to examine whether negative attachment cognitions interact specifically with parental depressive symptoms to predict child symptoms or whether they also interact with other forms of parental symptomatology (i.e., anxious symptoms). Future research should assess a broader range of symptoms (e.g., anxiety) to investigate issues pertaining to specificity at both the level of dependent (i.e., child symptoms) and predictor (i.e., parent symptoms) variables.

CONCLUSION

In sum, the results of the current study suggest that Bowlby’s (1980) attachment theory may prove to be a useful framework for understanding individual differences in vulnerability to depressive symptoms in children of affectively ill parents. As our study examined only one possible factor that may make youth more

susceptible to the deleterious impact of parental depression, future research is needed examining additional cognitive and interpersonal vulnerability factors. Identifying the factors that confer vulnerability to depression among high-risk youth is likely to be beneficial in guiding clinicians and researchers in designing effective treatment and prevention programs for such youth. Given the high level of risk for developing depression among such children, research in this area is desperately needed.

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